1110-151 Adverse Outcomes Prior to Hospital Discharge After Primary Stenting for Acute Myocardial Infarction Are Often Predictable, and Related to Correctable Technical Factors

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In the prospective PAMI Stent Pilot Trial, stenting was performed in 240 consecutive pts with AMI and eligible vessels at 9 sites (98% success rate). Independent core lab angiographic analysis was performed (Wash. Hosp. Center). As previously reported, in-hospital adverse events were infrequent, including death (0.8%), reinfarction (1.7%), recurrent ischemia (3.8%), and target vessel revascularization (CABG or re-PTCA, 4.6%); the composite of any of these events occurred in 15 (6.3%) patients. The factors predictive of any adverse event after primary stenting were examined from 20 clinical, 24 angiographic, and 7 procedural variables.

Results: From these 51 variables, only 2 correlates of pre-discharge adverse events were identified: stent implantation pressure < 18 atm., and the presence of a peri-stent dissection grade type B by core lab analysis. Adverse events occurred in 10.4% of pts with stents implanted at ≥ 18 atm., vs. 3.8% of pts stented at < 18 atm. ($p < 0.05$). Adverse events occurred in 20.4% of pts when a peri-stent ≥ type B dissection was present at the end of the procedure, vs. 5.3% if no dissection was present ($p = 0.02$). Adverse events occurred in only 2.9% of pts with neither of these 2 factors, vs. 11.5% with one or both ($p = 0.009$). All other important variables were unrelated to early adverse events including pt age, gender, diabetes, LVEF, number of stents implanted, stent type, use of ReoPro, reference vessel diameter, final MLD, and presence of thrombus pre or post stenting.

Conclusions: In-hospital adverse events are infrequent after primary infarct stenting, but are often predictable, and related to correctable technical factors. The routine use of ≥ 18 atm. of pressure to ensure complete stent expansion, and treatment of peri-stent dissection with additional stents to cover all margin tears may further improve the safety profile of primary stenting in AMI.

1110-152 Lack of Relationship Between the Time to Reperfusion and Short-term Mortality After Primary Infarct Angioplasty

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Studies of thrombolytic therapy have shown that in-hospital and 30 day mortality increases as time to treatment is prolonged. To explore whether the same relationship exists after primary PTCA, the effect of the time from symptom onset to PTCA on short-term mortality was examined by pooling 3 large primary PTCA databases: 1) The PAMI-2 trial (982 AMI PTCAs from 34 centers), 2) The Mid America Heart Institute (2098 AMI PTCAs), and 3) Moses Cone Hospital (1352 AMI PTCAs). In total, primary PTCA was performed in 4432 pts, 284 of whom (6%) presented in cardiogenic shock. The mean age was 60 ± 12 years, 28% were female, and 15% had diabetes. Short-term mortality occurred in 6.9% of pts, 45.4% of shock pts died vs. 4.2% without shock, $p < 0.0001$. Complete temporal data was available in 4362 patients (98.4%). Time to PTCA vs. short-term mortality appears in the Table:

Time to PTCA (hrs)	< 1	1–2	2–4	4–6	> 6	p
Non-shock pts – N	174	384	404	1189	931	–
Mortality (%)	3.2%	4.6%	3.8%	4.6%	4.5%	0.77
Shock pts – N	9	30	77	88	76	–
Mortality (%)	22.2%	41.9%	46.2%	46.1%	46.7%	0.69

The mean time from symptom onset to PTCA in non-shock patients who died vs. survived was 311 ± 264 vs. 327 ± 192 mins, $p = 0.77$, and in shock pts 319 ± 286 vs. 335 ± 305 mins, $p = 0.54$. In the most detailed database (PAMI-2), the only independent predictors of survival by multivariate analysis were young age, absence of 3VD, and establishment of TIMI-3 flow after PTCA, which was achieved in 93% of pts, independent of time to angioplasty.

Conclusions: These data suggest that whereas reperfusion by PTCA within 1 hour of symptom onset (possible in 4% of pts) may possibly improve early survival, mortality after 1 hour does not increase with delayed reperfusion, attesting to the fundamental importance of the open infarct artery for a favorable early prognosis.

1110-153 Axial Plaque Redistribution in Coronary Stenting: Implications for Reference Segment Measurements.

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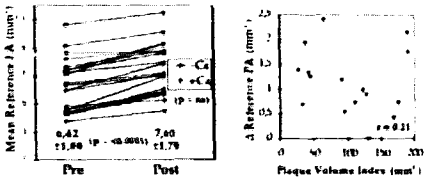
Background: Recent studies employing intravascular ultrasound (IVUS) have shown that balloon angioplasty is characterized by axial plaque redistribution from the lesion site to the adjacent artery segments. In coronary stenting, this mechanism may impact reference segment and relative stent expansion calculations as well as the degree of in-/outflow disease at stent edges. We determined the change in reference segment plaque area following coronary stenting (pre/post).

Methods: From our database, 20 stented patients were selected according to the following criteria: Pre- and post-interventional IVUS imaging performed with a motorized pullback device, 10 patients without IVUS signs of calcification, 10 patients with calcified lesions (> 90°, > 1 mm length). Standard IVUS parameters were measured in corresponding (pre/post) cross-sections in the reference segments (1–3 mm from the stent edge) to calculate the change (post-pre) in plaque area (PA). Mean lesion PA and lesion length before intervention were measured for calculation of a plaque volume index.



Results: A 1.18 mm² (20.65)-increase in average reference PA was seen upon the final IVUS pullback, independent of the presence of calcium or degree of preinterventional lesion plaque load.

Conclusion: There is a significant plaque redistribution to the reference segments impacting reference segment analysis.

1111 Role of Inflammation and Stress in Coronary Disease

Tuesday, March 31, 1998, Noon–2:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 1:00 p.m.–2:00 p.m.

1111-131 Low Frequency of Detectable Human Cytomegalovirus DNA in Coronary Atherosclerotic Lesions Obtained by Coronary Endatherectomy

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Background: Cytomegalovirus (CMV) infection has been implicated in the development of atherosclerosis and restenosis after coronary angioplasty. Previous studies demonstrated CMV in coronary specimen obtained by directional coronary atherectomy (DCA) and were limited to small sample sizes possibly underestimating the frequency of CMV-DNA within atherosclerotic lesions.

Methods: The presence of CMV-DNA in primary coronary lesions was assessed in coronary endatherectomy specimen obtained during successful aortocoronary bypass (ACB) surgery in 28 patients (23 men, 5 women, mean age 62 ± 9 yrs) with severe coronay artery disease (CAD). DNA from large coronary samples (5–30 mm long) was extracted using the QIA-Tissue-Kit (QIAGEN, Germany). Amplification of CMV target DNA sequences was carried out by using Taq polymerase (Boehringer, Germany). The PCR products were further specified by a novel liquid hybridisation assay called PCR-enzyme-linked immunosorbent assay.

Results: Positive PCR for CMV-DNA was found in 2/28 samples (7%) from coronary endatherectomy samples obtained during ACB surgery. Additional PCR-enzyme-linked immunosorbent assay specified all PCR-positive samples.

Conclusions: The low frequency of detectable human cytomegalovirus DNA in coronary atherosclerotic lesions (7%) does not support the hypothesis that a latent CMV infection of coronary arteries is a major pathogenetic factor for the development of CAD.